excessive free water loss, excessive sodium intake or diminished free water intake. The last mechanism seems the explanation in this and the four other reported cases.

All five infants shared common features: they were emaciated, less than 1 month of age and exclusively breast-fed-that is, with no water or formula supplementation (Table 1). Three of the infants were "sleepy," difficult to keep awake for feeding and content to starve uncomplainingly, a problem to which attention has recently been drawn.⁵ One mother was noted to be upset by her difficulty in arousing her baby, but was reassured by family members that this was normal.² In the three cases where maternal parity was described, all mothers were primiparous. This suggests a lack of familiarity with normal newborn eating and sleeping patterns, and may explain why these mothers delayed seeking medical attention.

Although three of the mothers appeared to have diminished milk production, an expected finding in the face of diminished demand, only one was reported as perceiving her milk production to be inadequate.3 Each had an unexplained increase in milk sodium concentration in comparison to a mean of 7±2 mEq per liter reported by Macy⁶ for women 15 days postpartum. In fact, these values were even high for women during the first five days postpartum when the mean sodium concentration is significantly higher (21 ± 5 mEq per liter).7 This last association is an intriguing but unlikely explanation for the hypernatremia. The infants' diminished intake, suggested by the emaciation and degree of dehydration, makes it improbable that they ingested a high amount of sodium. The cause of the increased milk sodium concentration remains uncertain.

Without any evidence of excessive free water loss, the explanation for the hypernatremia in these five infants is diminished free water intake. The combination of large insensible water losses diminished renal concentrating capacity makes newborn infants susceptible to water depletion. Even nursing infants who have low obligatory urinary water loss (because of breast milk's low solute content) can become water depleted when intake of breast milk decreases to the extent that the renal concentrating capacity is exceeded.

Comment

The five cases of hypernatremia in breast-fed newborns emphasize the importance of frequent follow-up of newborn infants. Careful attention must be paid to the adequacy of their weight gain. Although most babies cry when they are hungry, health professionals who care for newborns must be aware that otherwise normal babies may be content to nurse infrequently, and thereby fail to thrive and, in this context, become hypernatremic. With adequate instruction of mothers and proper care of their breast-fed infants, this potentially damaging condition⁸ can be prevented.

REFERENCES

- 1. Nutrition Committee of the Canadian Paediatric Society and the Committee on Nutrition of the American Academy of Pediatrics: Breast feeding—A commentary in celebration of the International Year of the Child, 1979. Pediatrics 62:591-601, 1978
- 2. Clarke TA, Markovian M, Griswold W, et al: Hypernatremic dehydration resulting from inadequate breast-feeding. Pediatrics 63:931-932, 1979
- 3. Anand SK, Sandborg C, Robinson RG, et al: Neonatal hypernatremia associated with elevated sodium concentration of breast milk. J Pediatr 96:66-68, 1980

 4. Arboit JM, Gildengers E: Breast-feeding and hypernatremia. (Editorial Correspondence) J Pediatr 97:335-336, 1980
- 5. Evans TJ, Davies DP: Failure to thrive at the breast: An old problem revisited. Arch Dis Child 52:974-975, 1977
- 6. Macy IG: Composition of human colostrum and milk. Am J Dis Child 78:589-603, 1949
- 7. Ansell C, Moore A, Barrie H: Electrolyte and pH changes in human milk. Pediatr Res 11:1177-1179, 1977
- 8. McCauley D, Watson M: Hypernatremia in infants as a cause of brain damage. Arch Dis Child 42:485-491, 1967

Refer to: Smith CI, Juttner HU, Redeker AG: Bile duct stricture shown during transhepatic cholangiography by cholecystokinin. West J Med 135:55-57, Jul 1981

Bile Duct Stricture Shown **During Transhepatic** Cholangiography by Use of Cholecystokinin

COLEMAN I. SMITH, MD Stanford, California

HANS U. JUTTNER, MD ALLAN G. REDEKER, MD Downey, California

THE HORMONE cholecystokinin (CCK) has been used to show gallbladder contraction during cholecystography (following oral ingestion of contrast material). We report the case of a patient with a stricture of the common bile duct in whom CCK was used during transhepatic cholangiography

From Liver Service, Department of Medicine and Department of Radiology, University of Southern California School of Medicine, Rancho Los Amigos Hospital, Downey, California Dr. Smith is now affiliated with Stanford University Medical Center, Stanford

Submitted June 19, 1980.

Reprint requests to: Allan G. Redeker, MD, Rancho Los Amigos Hospital, Liver Unit, 1100 Building—Room 53, Downey, CA 90242.

ABBREVIATIONS USED IN TEXT CCK = cholecystokinin THC = transhepatic cholangiography

(THC) to cause gallbladder contraction, which facilitated the outline of the biliary stricture.

Report of a Case

A 40-year-old black man was admitted to the Liver Unit, Rancho Los Amigos Hospital in December 1979 because of recurrence of abdominal pain and jaundice. The patient had been alcoholic for ten years and had had multiple bouts of acute pancreatitis. He was first noted to be jaundiced in October 1979 following one of these episodes. On that occasion, an endoscopic retrograde cholangiopancreatogram showed changes in the pancreatic ducts consistent with chronic pancreatitis and a stricture in the intrapancreatic portion of the common bile duct. The jaundice resolved and the patient was temporarily lost to follow-up after discharge from the hospital. On admission to hospital on December 20, he presented with a similar history and stated that he had continued to take alcohol. The only abnormalities found on physical examination were scleral icterus and a liver edge that was just palpable in the right costal margin on deep inspiration. Laboratory test results included a serum albumin of 3.9 grams per dl, serum alkaline phosphatase 11.3 Bessey-Lowry units (normal range 0.8 to 2.3), total bilirubin 10.6 grams per dl, serum aspartate aminotransferase (serum glutamic oxaloacetic transaminase) 524 IU per liter, serum alanine aminotransferase (serum glutamic pyruvic transaminase) 224 IU per liter, prothrombin time 100 percent of control value, serum amylase 1,870 U per ml, hemoglobin 15.2 grams per dl and leukocyte count 10,500 per cu mm, with a normal differential count.

An ultrasound examination of the liver and gallbladder showed evidence of dilated intrahepatic and extrahepatic ducts but no evidence of cholelithiasis. A percutaneous THC was done, using a 23-gauge Chiba needle. The needle first entered a right-sided bile duct. After aspirating about 30 ml of green bile, contrast material was injected. The right intrahepatic ducts were not dilated, but the left-sided ducts as well as the main hepatic and proximal common bile ducts were dilated. The gallbladder was considerably elongated and filled with contrast material. However, there was an

abrupt stop of the contrast material at the level of the suprapancreatic portion of the common bile duct, and a nipple-like deformity was noted (Figure 1). For ten minutes repeated attempts were made to place the patient in the upright position; however, no dye passed beyond the suprapancreatic portion of the common bile duct.

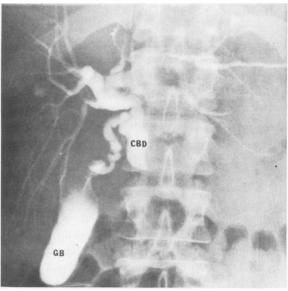


Figure 1.—Film of contrast material in bile duct and gallbladder (GB) before administration of cholecystokinin, showing what appears to be complete obstruction of the common bile duct (CB). This film was taken while the patient was in the upright position, after removal of the needle.

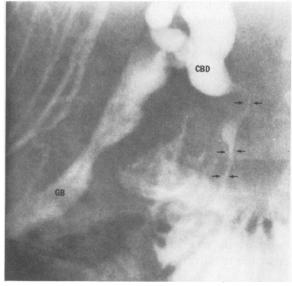


Figure 2.—Film taken three minutes after administration of cholecystokinin, showing substantial contraction of the gallbadder (GB). The contrast material outlines the stricture of the common bile duct (CBD) (arrows), with spillage into the duodenum.

Within two minutes following intravenous injection of 1.4 μ g of Kinevac (an active synthetic Cterminal octapeptide of CCK), there was substantial contraction of the gallbladder with passage of the dye into the duodenum and outlining of the strictured area of the common bile duct (Figure 2). There were no gallstones in the entire biliary system.

The patient tolerated the procedure with only a short episode of cramping abdominal pain. All laboratory test results returned to normal ranges within three weeks and the patient was discharged from the hospital as he declined any therapy for his bile duct problem.

Discussion

Transhepatic cholangiography is a useful procedure in the investigation of obstructive jaundice. The technique we used was similar to that described by Okuda and co-workers, in which a 23gauge Chiba needle is inserted percutaneously into an intrahepatic bile duct from an entry point in the right flank. In incomplete obstruction of the biliary system, placing the patient in the upright position often allows the contrast material to pass the obstruction and reach the small intestine. In this case, however, such a maneuver was unsuccessful. Therefore, CCK was used to cause contraction of the contrast-filled gallbladder which it was hoped would force the contrast material through the obstruction and allow it to reach the small intestine, thereby outlining the stricture. As shown in Figures 1 and 2, cck caused a very prompt contraction and the contrast material clearly outlined the stricture. CCK has been used in radiology for many years to investigate gallbladder contraction during cholecystography,2 but we are unaware of its use as described above. CCK may be a safe and useful adjunct in carrying out THC in selected circumstances. It can be useful in outlining a stricture in the common bile duct when the obstruction is incomplete and is below the entry of the cystic duct and when there are no small stones in the gallbladder that might be forced into the common bile duct. Presumably, this technique is of use only in gallbladders which are capable of contraction, as CCK does not appear to have an action on the bile duct.3

Summary

A stricture of the common bile duct developed in a 40-year-old man due to recurrent chronic alcoholic pancreatitis. The stricture was able to be visualized during transhepatic cholangiography following injection of cholecystokinin, which caused contraction of the gallbladder (that was filled with contrast material) and forced the material through the strictured area.

REFERENCES

- 1. Okuda K, Tanikawa K, Emura T, et al: Nonsurgical, transhepatic percutaneous cholangiography: Diagnostic significance in medical problems of the liver. Am J Dig Dis 19:21-36, 1974
- 2. Berk RN: Oral cholecystography in cirrhosis, *In* Berk RN, Clemett AR (Eds): Radiology of the Gallbladder and Bile Ducts. Philadelphia, WB Saunders Company, 1977, pp 129-136
- 3. Ludwick JR, Bass P: Contractile and electrical activity of the extrahepatic biliary tract and duodenum. Surg Gynecol Obstet 124:536-546, 1967

Refer to: Hickman DM, Frey CF, Carson JW: Adenocarcinoma arising in gastric heterotopic pancreas. West J Med 135: 57-62, Jul 1981

Adenocarcinoma Arising in Gastric Heterotopic Pancreas

DONN M. HICKMAN, MD CHARLES F. FREY, MD Sacramento, California

JAY W. CARSON, MD Martinez, California

HETEROTOPIC PANCREATIC TISSUE may be found anywhere from the distal end of the esophagus to the colon in the alimentary canal. Its location outside the alimentary canal also has been reported.1-4 Symptoms associated with heterotopic pancreas are uncommon, but when present are usually associated with a gastric site. In this location, symptoms resemble those of peptic ulcer or pyloric obstruction.2,5,7-10 The spectrum of pathological changes characteristic of the main gland including, rarely, pancreatic cancer is shared by heterotopic pancreas.^{2,3,6,11-21} This paper reports a case of gastric heterotopic pancreas giving rise to a nonislet cell pancreatic carcinoma. A discussion of other reported cases of pancreatic cancer thought to have arisen from gastric heterotopic pancreas is given and a plan for surgical management is recommended.

From the Departments of Surgery and Surgical Pathology, Veterans Administration Medical Center, Martinez, and the Department of Surgery, University of California, Davis, School of Medicine.

Submitted July 9, 1980.

Reprint requests to: Charles F. Frey, MD, UCD Professional Building, Room 257, 4301 X Street, Sacramento, CA 95817.